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(*Continued.*)

BROWN-SEQUARD PARALYSIS, WITH REPORT OF A CASE.

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ON November 7, 1895, Charles Gross, a negro laborer, 29 years of age, was admitted into the City Hospital suffering from the effects of a gunshot wound. Examination of the patient showed a wound made evidently by a small bullet, just below the angle of the jaw on the left side. The patient was suffering somewhat from shock when admitted. Examination of the chest showed the heart sounds to be clear and of normal relative intensity; the pulse, however, was regularly intermittent, dropping every third beat. The lungs were clear throughout, though the left chest seemed to lag a little. The left arm was completely paralyzed, and the left leg partially paralyzed, the patient being unable to raise it from the table, though he could move it slightly. A probe passed into the wound followed the track of the bullet backward for about two inches and then lost the course. It is to be noted that the man was in a stooping position when shot.

A careful examination of the patient the next day revealed the following: The general appearance of the man is good; he is unusually well-developed and muscular, and says he has always been perfectly healthy, with the exception of what from his account must have been a bubo dating back two years. Family history is good. There was no loss of consciousness after the injury, though, he says, he felt considerably dazed. He fell immediately upon being shot, and was not able to get up from the ground without assistance.

There is no loss of power on the right side. On the left side, there is loss of power complete in the arm and almost complete in the leg. He cannot raise the leg, but can move it about slightly on the bed. Reflexes superficial and deep are apparently unaltered. On the right side there was impairment of tactile sensibility, and complete loss of pain sense and temperature sense, from a point a little below the clavicle down. The accompanying charts show the sensory disturbances, as regards pain and temperature sense.

Muscular sense was greatly impaired in the left arm; slightly in the leg. There was no disturbance of bladder or rectum.

On Nov. 19th, twelve days after the injury, the patient managed to get up, though he dragged the left leg in walking. The left arm is still completely paralyzed. On the left side there is greatly exaggerated knee reflex and ankle clonus. From the chart of this date it will be seen that the sensory disturbances are lessening in extent. The notes made Nov. 28th show that patient can walk but still drags the left leg and that the paralysis of the arm has not improved. The increased knee-jerk with ankle clonus continues in the left leg, and there is a slight ankle clonus observable in right foot. The sensory disturbances have still further cleared up.

Dec. 8th. Sensation is almost normal. Has slight movement in the fingers of the left hand. Left knee-jerk greatly exaggerated and ankle clonus marked on this side. There is a barely perceptible ankle clonus on the right side. The patient left the hospital Dec. 10th.

May 23d. The patient was examined on this date, nearly seven months after the receipt of the injury. He has regained perfect use of the left leg. The left arm is still markedly affected; he is unable to place the hand on the back of the neck, or at least can perform this movement very slowly and with great difficulty. He says he is unable to lift any heavy weight with the left arm. The dynamometer registers 105 on the right side and only 30 on the left. There is decided atrophy of the shoulder and arm muscles.

	RIGHT ARM.	LEFT ARM.
Wrist,	16 cm.	16 cm.
Forearm uncontracted,	26 "	24 "
Forearm contracted,	28 "	26 "
Biceps contracted,	32 "	25 "

There is greatly diminished reaction to both currents in left arm, and slightly diminished reaction in left leg. The knee reflex is greatly exaggerated on the left side, and there is marked ankle clonus. There is no sensory disturbance on this side, and tests as to muscular sense showed it to be normal.

On the right side, the side opposite the lesion, there was no loss of power, no atrophy of muscles, and the reflexes were normal. Tactile sensibility appeared to be normal, except perhaps on the back of the forearm. In this situation there seemed also to be some analgesia. These disturbances were, however, trifling. Over the whole right side there was absolute loss of temperature sense. He was entirely unable to distinguish between a test tube containing hot water and one filled with ice water. This was tested with the greatest care. This is an extremely interesting point since the temperature sense had nearly entirely returned when he left the hospital Dec. 10th.

The symptoms observed in this case, viz: almost complete paralysis on the side of the lesion with loss of muscular sense, and loss of pain and temperature sense on the opposite side, suggested, of course, Brown-Séquard paralysis, the lesion presumably being about the level of the fourth cervical nerve.

Brown-Séquard, in 1863, from a limited number of experiments on animals, but chiefly from the study of the records of clinical cases, announced that a lesion involving one lateral half of the spinal cord gave rise to certain definite and constant symptoms. His conclusions are as follows (*Journal de la Physiologie*, t. vi., 1863): " In the human subject after a lesion destroying a small portion of one lateral half of the spinal cord, the following phenomena are observed: 1st. On the side of the lesion paralysis of voluntary motion, hyperæsthesia to tactile, pain and temperature senses, a limited zone of anæsthesia, and below this hyperæsthesia and elevation of temperature. On the side opposite the lesion, preservation of voluntary motion and muscular sense, complete anæsthesia, and a limited zone of hyperæsthesia above the anæsthetic parts." The persistence of sensibility of all kinds on the side of the lesion and the absolute loss of the corresponding senses on the opposite side were taken as incontestible proofs of an entire crossing of all the sensory fibres with the exception of those conveying muscular sense. No experiments of any importance bearing on the subject were made until those of Ferrier in 1886. ("Functions of the Brain," p. 51). From a number of experiments on the monkey, Ferrier came to much the same conclusions as those announced by Brown-Séquard, except that he laid stress on the fact that in his experiments muscular sense was lost on the side opposite the section, and also that in monkeys there was not noted the hyperæsthesia on the side of section which seemed to be the rule in the human subject. Ferrier makes in this connection the important observation that it is probable that the sensory paths are not so exclusively crossed in many of the lower animals as they seem to be in man and the monkey, and that the paths of voluntary motor impulses are not rigorously confined to one side of the cord. Erb (Ziemssen's Encyclopædia, vol. xiii.) calls attention to the girdle sensation felt at the limit of the lesion, to the occasional disturbances of bladder and rectum, to the fact that muscular atrophy may occur, and to certain trophic manifestations. Charcot (Œuvres Completes, t. I. and II.) calls attention to the frequency of muscular atrophy and to the trophic changes. All the writers on the subject followed Brown-Séquard in his explanation of the clinical observations, but it is remarkable what differences are to be observed in regard to muscular atrophy, hyperæsthesia, muscular sense, tendon reflexes and other symptoms. When experiments upon animals began to be performed under strict antiseptic precautions, it became evident that Brown Séquard's results could not be accepted *in toto.* Turner in an interesting and valuable paper (*Brain*, vol. xlv., 1891) reviews the original cases of Brown-Séquard, and adds some important experimental work upon monkeys. He concludes from the study of these and other cases that from the purely clinical standpoint it would seem that (1) Tactile sensibility was present on the paralyzed side, and in the majority of cases there was hyperæsthesia to all forms of sensation. In most cases this was true of both arm and leg when the lesion was cervical. Pain and temperature sense followed the same rule. In regard to muscular sense there was great difficulty in arriving at any definite conclusions, since in many instances it was not tested. Brown-Séquard, from his study of the cases that he collected, believed that this sense was absent on the paralyzed side. (2) On the side opposite the lesion evidence pointed to complete anæsthesia to all forms of sensation except,perhaps, muscular sense, concerning which, as was stated above, there is great discrepancy in the recorded cases.

In studying these cases after a lapse of months or years it was seen that recovery was very irregular, but generally the paralysis persisted much longer than the sensory disturbances. The organic functions were sometimes temporarily interfered with, and the reflexes at first diminished on the paralyzed side, later became greatly exaggerated. From his experiments on monkeys Dr. Turner concluded that the sensory fibres decussated shortly after their entrance into the cord. This he thought held good in the monkey only for the hinder extremities. His experiments upon the cervical region seemed to indicate that sensibility was not entirely abolished on the non paralyzed side.

Coming down to 1894 we find Brown-Séquard renouncing in great part the views he had held so long. It was a splendid example, and one worthy of emulation, when this true scientist abandoned a theory that he had held for so many years, and one that had shed such lustre upon his name, because certain experiments, partly his own, partly the work of others, failed to completely bear out his views. In the *Archives de Physiologie*, Jan., 1894, he refers to an experiment of his own in which it was shown that the anæsthesia produced by a hemisection of the cord in the cervical region is immediatly replaced by hyperæsthesia after a hemisection of the dorsal cord, and at the same time the hyperæsthesia caused by the first lesion is replaced by anæsthesia. In addition to this he adduces the fact that a simple pricking of the posterior column of the dorsal cord often suffices to produce the exact symptoms of a hemisection. In the same manner section of the posterior nerve roots in the upper dorsal region on one side will produce hyperæsthesia on the corresponding side with anæsthesia on the opposite side. He also cites the experiments upon animals, in which, after hemisection of the cord in the dorsal region, stretching of the sciatic nerve on the side opposite the section will cause a disappearance of the anæsthesia. In the same paper he points out the great difference in function between the cords of the lower animals and man, and concludes that whatever be the cause of the symptoms clinically, the fact remains that in man a hemisection will produce a crossed anæsthesia, and refers to his 80 collected cases as proof. The only explanation he suggests, admitting that the phenomena cannot be explained by direct interference with conduction, is that the section or injury of the cord must act as an excitor of inhibition.

Mott (Proc. Philosoph. Soc., Jan. 1, 1891) concludes from his experiments on monkeys, that after hemisection there is no very well marked anæsthesia on the side opposite the section, and in some instances there is distinct analgesia on the side of section. Schiff's clamp-test showed that pressure sense was present on the non-paralyzed side while absent on the paralyzed side. The evidence from Gowers' and Horsley's collection of cases of tumor of the cord points to the non-decussation of the sensory impulses in the cord. The experiments of Horsley and Gotch accord with these views. The same author (Mott) in a later paper (Brain, vol. xviii., 1895) gives the results of a series of experiments on monkeys, in which he cut the lumbo-sacral roots on one side, and stained the sections after the Marchi method. In brief, these experiments may be said to show that "the degenerated fibres do not pass into the opposite posterior column, nor do they enter any of the long ascending tracts of the anterior or lateral columns." The author would explain the occurrence of degenerated fibres in the crossed afferent tracts as due to vascular changes in the gray matter of the posterior horn. He also agrees with Edinger that "there is a path for afferent impressions from the posterior roots through cells of the gray matter, and by fibres from the latter which decussate in the anterior commissure."

These fibres are supposed to ascend the cord in the antero-lateral tracts to the fillet. As Mott says, "we seem to be coming back to the views of Schiff, that the gray matter conducts painful sensations and heat and cold, whilst the posterior columns convey the tactile and muscular sense impressions." It may be said that in general the researches of Ramon y Cajal and other modern neuro-histologists, favor the idea that the great proportion of the fibres subserving the function of sensory conduction pass upon the same side of the spinal cord that they enter, and decussate in the fillet.

The conclusions to be drawn from the foregoing clinical and experimental observations are as follows:

1st. It is doubtful whether we can compare the sensory conduction of the spinal cord of the lower animals, even the monkey with man.

2d. It is probable that the main sensory pathway is uncrossed in the spinal cord.

3d The clinical cases in man showing permanent or long continued sensory disturbances on the side opposite the supposed lesion are probably due to an injury that involves more than half the cord.

4th. The temporary disturbances on the side opposite the lesion are probably to be explained on the theory of inhibition.